OZONE EFFECTS ON HEART RATE AND REPOLARIZATION PARAMETERS IN POTENTIALLY SUSCEPTIBLE INDIVIDUALS

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Background and Aims. There is evidence for an association between elevated ozone levels and cardiovascular morbidity and mortality. However, knowledge about ozone effects on electrocardiogram (ECG) parameters is very limited. We investigated the effects of ozone on heart rate (HR) and repolarization parameters in potentially susceptible populations.

Methods. Between March 2007 and December 2008, 363 ECG recordings comprising more than 2000 1h-intervals were measured in 64 individuals with type 2 diabetes mellitus or impaired glucose tolerance and in 46 healthy individuals with a potential genetic predisposition on the detoxification pathways from Augsburg, Germany. Ambient ozone and meteorological data were acquired from a fixed monitoring site on an hourly basis. Associations between 1h-averages of ambient ozone and HR, Bazett-corrected QT-interval (QTc), T-wave amplitude (Tamp) and T-wave complexity (Tcomp) were analyzed using additive mixed models adjusted for long-term time-trend and meteorological variables. A variable indicating both the season and whereabouts of the participants during the 1h-ECG recordings (summer and outdoors vs. winter or indoors) was used to assess potential modification of the ozone effect.

Results. Increases in ozone by 20μg/m³ (£10ppb) led to concurrent and 1h–4h delayed increases in HR by 0.5-0.7% in all participants. These effects were stronger (1.0-1.2%) when the participants were outdoors during summer. Furthermore, we detected in all participants a concurrent and 1h-lagged T-wave flattening of -1.31% [95%-confidence interval:-2.19;-0.42%] and -1.32% [-2.19;-0.45%], respectively. Correspondingly, 1h- and 2h-lagged increases in Tcomp by 2.12% [0.81;3.52] and 1.89% [0.55;3.26%] were associated with elevated ozone levels, respectively. However, no effects could be observed for QTc. In general, ozone effects were more pronounced in individuals with metabolic disorders.

Conclusion. Our observed findings might be a mechanistic link between elevated ozone levels and reported adverse cardiovascular outcomes.